Stated Meeting, February 24, 1896.

Vice President, Dr. C. W. BURR, in the chair.

Dr. FRANCIS X. DERCUM read a paper on

TROPHIC LESIONS OF THE FEET TOGETHER WITH SYMPTOMS SUGGESTING GLIOSIS OF THE CORD.

Dr. F. SAVARY PEARCE reported

A CASE OF CHRONIC PROGRESSIVE PARALYSIS DUE TO PRIMARY MUSCULAR DYSTROPHY; AUTOPSY, HISTOLOGICAL STUDY.

(ABSTRACT.)

Mrs. F., 59 years of age, seen by Dr. S. Weir Mitchell first, in November, 1886. At that time she had had muscle paresis for eighteen years. There was no hereditary history of nervous disease, and no alcoholic, metallic or other toxic or infectious process as an ætiological possibility in the case. Of four children, one died of phthisis florida at the age of twenty-six years. The present trouble was attributed to a fall in 1866 (at the age of thirty-two years); for not long after the patient noticed a weakness of third and fourth fingers both hands, due to paresis of extensors of forearms. After several years of this insidious onset, double wrist-drop developed. The foot and leg extensors now failed in power and she constantly caught her toes in rugs about the room from inability to elevate the foot. There was no pain or tenderness of the parts involved. Locomotion gradually became more and more difficult. There was a distinct swagger in her gait. The flexor muscles did not become markedly involved until nine years later (at age of forty-one years). Then she had become very helpless.

Since that time there had been gradual increasing palsy in the flexor muscles of the forearms and arms; with more rapid loss of power in the flexors of thighs and in the calves of the legs. Knee-jerks and elbow-jerks absent. But little muscular response on striking a firm blow. The order of fiexor muscle paralysis had thus been the reverse of the antedating failure of the extensor muscles.

There was no fibrillary contractions; no anæsthia existed. There was great quantitative and little qualitative change in the electrical response of the muscles.

The woman gradually lost power in the hands so that she could not make shaded letters in writing. Finally her ability to write at all was almost completely lost, and she had to be fed like a child. She could stand alone, however, for a short time when placed on her feet. This was probably made possible by the knees being held in extreme extension permitting the support to be almost entirely skeletal. The slightest push would occasion a fall. The patient complained of nothing during the prolonged illness. She was always bright and cheerful.

Intellect remained remarkably good until the last. Memory for classifying events was extremely acute. Speech and deglutition were unaffected. There was never any marked atrophy anywhere; on the contrary, the muscles generally were full and firm on palpation; all out of proportion to her extreme weakness; striking the parts produced transient redness due to vasomotor weakness.

The muscle palsy had thus existed for over twenty-seven years. On February 19, 1895, the patient took a chill and died of double lobar pneumonia March 7, 1895. Autopsy eighteen hours later. No scars or deformities were noted excepting slight atrophy of the hand interossei muscles; and rigid extension of ankles.

Much bright yellow fat infiltrated the abdominal walls, most all muscles of the thorax and abdomen having undergone fatty metamorphosis. Lungs presented the signs of recent lobar pneumonia limited to both bases. Heart was in an advanced state of fatty degeneration, only an area the size of a twenty-five cent piece being evident to the naked eye. Diaphragm had also become converted largely into fat.

Other organs presented no pathological signs. Biceps muscles of the arms were of fair size, but really masses of fat. Recti femori on the right showed the same advanced fatty change. Biceps and other hamstring muscles presented the same process to a less degree.

Spinal cord showed no macroscopic changes. The cord, sciatic, external and internal popliteal nerves, and portions of the muscles spoken of were preserved for study.